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Original Article

Time course of vascular dysfunction of brachial artery after trans-radial access for coronary angiography

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ABSTRACT

Background: Prior studies have demonstrated that brachial artery endothelial dysfunction will occur after transradial cardiac catheterisation for diagnostic coronary angiography. However, the duration of this endothelial dysfunction was unknown.

Methods: A total of 74 patients, who are undergoing coronary angiogram through transradial route were included. Using high-resolution vascular ultrasound endothelium-dependent flow mediated dilatation (FMD) and endothelium-independent dilatation (NMD) after administration of sublingual nitroglycerin of ipsilateral brachial artery (IBA) were measured before, 6 h, 24 h, 1 week and 1 month after catheterisation. The left-sided brachial artery (BA) served as a control.

Results: Base line FMD was 13.8 ± 5.6, 14.8 ± 5.2 in the right and left brachial arteries ($p = 0.26$). FMD was significantly decreased in the right intervention arm, when compared to control at 6 h and 24 h (6.8 ± 6.0, 14.5 ± 5.0, p value 0.000), (7.9 ± 7.4, 15.3 ± 4.8, p value 0.000), at 1 week BA FMD recovered to baseline (12.6 ± 7.5, 15.7 ± 4.5, p value 0.06) whereas at 1 month test BA FMD was not statistically significant from baseline test FMD (16.5 ± 5.5, 16.1 ± 4.7, p value 0.07). In contrast both NMD response and FMD/NMD ratio were unaffected by the intervention in the control arm. K–M curve showing the probability of recovery of BA FMD after transradial intervention is 90% at 30 days.

Conclusion: Trans-radial cardiac catheterisation leads to vascular endothelial dysfunction of the IBA with recovery of the vascular dysfunction by 1-week post-procedure.

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1. Introduction

In recent years flow mediated dilatation (FMD) has become a popular technique in cardiovascular medicine and clinical physiology as evidence has occurred that depressed FMD is an independent prognostic index of incident and recurrent cardiovascular events, which adds predictive value to the established risk factor.[1–5]

The FMD measurement has gained growing interest as several studies indicated that a decreased FMD response predicts arterial disease progression with intimal thickening and increased cardiovascular mortality.[6,7] Several cardiovascular risk factors have been shown to lead to acute and chronic FMD impairment.[6,8] Physical damage to the vascular endothelium might also be a cause of functional impairment and might lead to arterial disease.[9] In the context of BA FMD this is particularly important because the transradial route to perform coronary angiograms and percutaneous coronary interventions is an emerging safe alternative to the femoral access due to its lower bleeding complications.[10,11] We noninvasively studied the duration and the effect of transradial diagnostic cardiac catheterisation on endothelium-dependent and endothelium-independent vasodilatation of the BA in selected patients.

2. Methods

2.1. Study patients

Seventy-four (74) consecutive in-patients with a normal Allen's test, who underwent transradial intervention (TRI) via the radial artery (RA) from the department of cardiology were enrolled in this study during the period of May 2013 to November 2013. Ethical committee approval and informed consent from each patient were obtained. Patients were excluded from the study if they had undergone previous radial cannulation or had an abnormal Allen's test result consistent with insufficient ulnar collateral supply. Other exclusion criteria were acute inflammation (C-reactive protein >0.5 mg/dl), malignancies, heart rhythm other than sinus rhythm, and heart failure New York Heart Association functional class III–IV.

2.2. Procedural protocol

The right arm was positioned beside the patient's body and the wrist was hyper extended. After local subcutaneous anaesthesia with 1% lidocaine, RA puncture was performed with a 21-gauge angiocatheter needle (Cordis Corporation, USA) and a 0.53 mm (0.021 in.) hydrophilic guide wire was inserted through the needle. Upon removal of the needle, an 11 cm 6-F sheath (Cordis Corporation, USA) was installed over the guide wire. After sheath insertion, all patients received 0.2 mg nitro-glycerine and the bolus of heparin (5000 IU for CAG) through the sheath. After transradial coronary procedure, the arterial sheath was removed immediately and haemostasis was achieved by RA compression.

2.3. Determination of vascular function with ultrasound

Clinical examination included blood pressure measurement, cardiovascular examination, and body-mass index. Biochemical assessment included fasting blood sugar and post-prandial blood sugar levels and comprehensive lipid profile. Plasma glucose and lipid estimation were done after an overnight fast of 12 h. The ultrasound method for measuring endothelium dependent and endothelium independent arterial dilatation was done as described previously. The brachial artery (BA) diameter was measured on B-mode ultrasound images with the use of a 7.5 MHz linear array transducer with image point HX Ultra sound Equipment (iE33 2-D Echo, Philips Ultrasound bothell, Washington, USA). The right BA was studied in all the subjects. BA endothelial function was studied after the subject had abstained from alcohol, caffeine, and smoking for 8 h. Scans were obtained with the subject at rest, during reactive hyperaemia and again at rest. The subjects were asked to lie quietly for at least 10 min before the first scan. The BA was scanned in longitudinal section 2 cm above the elbow and the centre of the artery was identified when the clearest picture of the anterior and posterior intimal layers was obtained. The transmit (focus) zone was set to the depth of the near wall, because of the greater difficulty in evaluating the ''M'' line (the interface between the media and adventitia) of the near wall as compared with that of the far wall. Depth and gain settings were set to optimise images of the interface between the lumen and the arterial wall and the images were magnified. Settings for operating the machine were not changed during the study.

When a satisfactory transducer position was found, the skin was marked and the arm was kept in the same position throughout the study. A resting scan was obtained. The arterial diameter was measured. Increased flow was then induced by the inflation of a sphygmomanometer cuff placed around the forearm (distal to the scanned part of the artery) to a pressure of 200 mmHg for 5 min followed by release. A second scan was performed continuously at 30 s, 60 s and 90 s after deflation of the cuff. The diameter of the artery was measured at the peak of R wave (corresponding to end diastole).

Flow-mediated dilatation was calculated, and the average results of the three observations recorded. Flow-mediated dilatation was presented as the percent change from baseline to hyperaemia. Severe endothelial dysfunction was defined as FMD <5.5%, as has been described. 15 min was allowed for the vessel recovery and then a further resting scan was performed, then sublingual glycerol-tri-nitrate (GTN-200mics puff) was administered and 4 min after the last scan was done. ECG was monitored throughout the scans and artery diameter measured at the peak of R wave (corresponding to end diastole). An average of 3 values was taken for each measurement. FMD and endothelium-independent dilatation (NMD) (nitrate mediated vasodilatation) was compared with that of control left arm.

2.4. Calculation of image analysis, calculation of vasodilatation and wall shear stress (WSS)

All diameter readings were taken at diastole, and flow velocity represents the mean angle-corrected Doppler flow velocity.